Lipid Replacement Therapy and Mitochondrial Damage in Alzheimer's Disease and Dementia

Professor Garth Nicolson explains how Lipid Replacement Therapy with NT Factor is an effective way to reduce chronic fatigue associated with neurodegenerative diseases and also reduces the effects of excess oxidative damage to our nerves and other cells

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Many diseases are associated with chronic fatigue, including neurodegenerative, neurobehavioral, respiratory, coronary, musculoskeletal, metabolic and gastrointestinal diseases as well as infections and cancer. In fact, chronic fatigue is the most common complaint of all patients seeking general medical care, and almost all patients with neurological diseases experience various degrees of fatigue along with their neurological symptoms.

Within the neurological diseases dementia is one of the most common conditions. Dementia is found in various degenerative neurological diseases and is characterized by loss of thinking, memory and other brain functions caused by damage to brain cells and brain cell death. The most common form of dementia, accounting for 50-80% of cases, is Alzheimer's Disease, named after the German psychiatrist Alois Alzheimer who first described it in 1906.

Alzheimer's Disease usually strikes older people (the majority are 65 and older), but in about 5% of cases it can affect people in their 40s. The early stages of Alzheimer's Disease are often confused with the changes that take place during normal aging. However, Alzheimer's Disease differs from the natural brain aging process in that it is a progressive brain disorder that gradually worsens with age and damages and eventually destroys brain cells, resulting in loss of memory, intellectual abilities and other brain functions [1].

Alzheimer's Disease is the sixth leading cause of death in the United States, affecting approximately 5.3 million people. Most Alzheimer's Disease patients can live for some time with their condition, often ranging from 5 to 20 years or more, depending on their age and other health conditions [2].

Alzheimer's is predicted to affect 1 in 85 people globally by the year 2050 [3].

The Symptoms and Characteristics of Alzheimer's Disease

Usually the earliest symptom of Alzheimer's is difficulty remembering newly acquired information. This occurs because Alzheimer's changes in the brain typically occur in the parts of the brain that affect learning and memory. However, as Alzheimer's advances or progresses through the brain, increasingly more severe symptoms appear, such as disorientation, mood and behavioral changes, increased confusion about specific events and their time and place, unfounded suspicions about family, friends, coworkers and health care personnel, and eventually difficulty speaking, swallowing and walking along with increasing fatigue [1].

What makes Alzheimer's Disease different from natural aging can be found at the cellular level. Alzheimer's Disease is characterized by loss of connections between nerve cells, inflammation and build up inside nerve cells of microscopic clumps of altered proteins called beta-amyloid proteins that can form plaques, twisted and tangled microscopic strands of filaments containing components called tau proteins and alterations in nerve synapses, the specialized signaling regions between nerve cells or the signals they send to other nerve cells [4].

Mitochondrial Membrane Function in Alzheimer's Disease

In each of our cells, energy is produced in the form of high-energy molecules in our mitochondria, small two-membrane separated organelles responsible for energy production. When our mitochondria are damaged, due to aging, disease and even pharmaceutical treatments, they do not produce enough high-energy molecules to keep cells functioning properly. The most common way in which mitochondria are impaired is by oxidative damage to mitochondrial membranes by cellular free-radicals called Reactive Oxidative Spcies or ROS. Excess ROS oxidize mitochondrial membrane lipids, making the mitochondrial membranes less capable of insulating the energy-producing part of the mitochondria, resulting in lowered production of high-energy molecules needed by the cell.

In Alzheimer's and other neurodegenerative diseases excess ROS and oxidation of membrane lipids are believed to play a central role in disease development [5]. The lipids in membranes, and specifically those found in nerve cells, are particularly susceptible to ROS and oxidative stress. This can result in the production of neurotoxic products as well as oxidized lipids that can affect the pathogenic processes that produce the altered beta-amyloid and tau proteins that produce Alzheimer's nerve cell plaques and tangled filaments. Such oxidative stress and ROS can damage nerve cell membranes in the synapses, the communication regions between nerve cells, can also be affected along with nerve cell mitochondria and cellular energy production [5].

In Alzheimer's and other neurodegenerative diseases there are processes that produce excess ROS and oxidative damage to lipids and other cellular components. For example, heavy metals and infections, found often in Alzheimer's patients and other neurodegenerative diseases can result in excess ROS and oxidative damage [6, 7].

Lipid Replacement Therapy and Neurodegenerative Diseases

Until recently the only treatments for Alzheimer's and other neurodegenerative diseases were pharmaceuticals that treated the production of beta-amyloid protein, heavy metals or by correcting general nutrition. At the Institute for Molecular Medicine research is undergoing on the use of immunotherapy to treat beta-amyloid deposition, anti-infectives to treat various infections found in neurodegenerative diseases and Lipid Replacement Therapy to protect nerve mitochondrial and cellular membranes (see www.immed.org).

Lipid Replacement Therapy with NT Factor® is an especially attractive, all-natural approach to reduce nerve cell membrane damage and reverse the effects of excess ROS damage to cellular lipids [8]. NT Factor® provides cells with the specific types of membrane lipids that can repair mitochondria and cell membranes and make them functional again. The uniqueness of NT Factor over other lipid supplements is that NTFactor's lipids are required by mitochondrial and other membranes for their function, and NT Factor lipids are protected from damage by ROS and other factors that damage most lipids before they even reach our cells.

Lipid Replacement Therapy with NT Factor is an effective way to reduce chronic fatigue associated with neurodegenerative diseases and also to reduce the effects of excess oxidative damage to our nerves and other cells.

References:

1. Förstl H, Kurz A. Clinical features of Alzheimer's disease. European Archives of Psychiatry and Clinical Neuroscience 1999; 249 (6): 288-290.

2. Mölsä PK, Marttila RJ, Rinne UK. Long-term survival and predictors of mortality in Alzheimer's disease and multi-infarct dementia. Acta Neurologica Scandanavica1995; 91 (3): 159-64.

3. Brookmeyer, R; Johnson, E; Ziegler-Graham, K; Arrighi, HM. Forecasting the global burden of Alzheimer's Disease. Alzheimer's and Dementia 2007; 3 (3): 186-91.

4. Tiraboschi P, Hansen LA, Thal LJ, Corey-Bloom J. The importance of neuritic plaques and tangles to the development and evolution of AD. Neurology 2004; 62 (11): 1984-9.

5. Axelsen PH, Komatsu H, Murray IV. Oxidative stress and cell membranes in the pathogenesis of Alzheimer's disease. Physiology (Bethesda) 2011; 26(1): 54-69.

6. Obulesu M, Venu R, Somashekhar R. Lipid peroxidation in Alzheimer's Disease: emphasis on metal-mediated neurotoxicity. Acta Neurologica Scandanavica 2011; Feb. 9 [Epub ahead of print].

7. Nicolson GL, Haier J. Role of chronic bacterial and viral infections in neurodegenerative, neurobehavioral, psychiatric, autoimmune and fatiguing illnesses: Part 1. British Journal of Medical Practitioners 2009; 2(4): 20-28.

8. Nicolson GL, Ellithrope R. Lipid replacement and antioxidant nutritional therapy for restoring mitochondrial function and reducing fatigue in chronic fatigue syndrome and other fatiguing illnesses. Journal of Chronic Fatigue Syndrome 2006; 13(1): 57-68.

About the Author:

Professor Garth L. Nicolson is the President, Chief Scientific Officer and Research Professor at the Institute for Molecular Medicine in Huntington Beach, California. Professor Nicolson has published over 600 medical and scientific papers, edited 16 books, and served on the Editorial Boards of 30 medical and scientific journals and Senior Editor of four journals. Professor Nicolson has won many awards, such as the Burroughs Wellcome Medal of the Royal Society of Medicine (United Kingdom), Stephen Paget Award of the Metastasis Research Society, the U. S. National Cancer Institute Outstanding Investigator Award, and the Innovative Medicine Award of Canada. He is also a Colonel (Honorary) of the U. S. Army Special Forces and a U. S. Navy SEAL (Honorary) for his work on Armed Forces and veterans' illnesses.

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